

Determination of Risk Factors and their Association with Certain Laboratory Tests of Nonalcoholic Fatty Liver Individuals in Sulaimani City/Kurdistan Region of Iraq

Hardy Hassan Rasul¹

Bakhtyar Kamal Talabani¹

¹ Community Health Department, College of Health and Medical Technology, Sulaimani Polytechnic University, Sulaimani, KGR, Iraq

Abstract

Background and Objectives:

Nonalcoholic fatty liver disease is caused by the accumulated high amount of fat in the liver independently of alcohol consumption. The aim of this study was to identify risk factors and their association with certain laboratory tests in nonalcoholic fatty liver patients.

Methods:

This case-control study was conducted from February 2022 to July 2022 in both Shar Hospital (public sector) and Baxshin Hospital (private sector). 148 Kurdish adult, their age 18 to 70, participated in the study. 148 participants were interviewed in a questionnaire; 74 peoples were of NAFLD (cases), and 74 were without liver disease (controls). SPSS was used to analysis the data.

Results:

A total of 148Kurdish adults were included in the study. Participants in control group had a higher intake of tea and vegetables compared to case group, was statistically significant. But smoking and physical activity had no statistically significant relationship with NAFLD between bot group ($P>0.05$). Patients with NAFLD had dyslipidemia, higher mean (s.triglyceride, LDL, and s.cholesterol levels, and lower mean HDL values) than those without NAFLD, and statistically significant differences ($P<0.05$).

Conclusions:

It was concluded that drinking tea and eating vegetables strongly correlate with NAFLD because they help the liver be protected from fatty liver changes.

Keywords:NAFLD, dietary pattern, Biochemical test, liver disease, Iraq.

1. Introduction

Nonalcoholic fatty liver disease (NAFLD) is a common type of liver disease in patients with metabolic disorders such as overweight, hyperglycemia, and hyperlipidemia[1].NAFLD is defined by hepatic steatosis (HS) of more than 5% in the absence of hepatocellular injury as hepatocyte ballooning[2].

Nonalcoholic fatty liver (NAFL) and nonalcoholic steatohepatitis (NASH), including liver inflammation, are two forms of this liver condition[2].NAFLD is safer than NASH because it

seldom develops into liver cirrhosis or NASH[2,3]. Severe outcomes, such as cirrhosis, hepatocellular cancer, liver failure, and cardiovascular problems, are possible if NAFLD progresses to NASH[3].

The prevalence of NAFLD in the general population might differ depending on the diagnosis methods used. Experts estimate that 10–35% of the population has NAFLD[4–6]. Increasing rates of obesity, changing dietary habits, and decreasing levels of physical activity have contributed to an increase in the prevalence of NAFLD in Eastern countries [7,8].

Insulin resistance (IR), a high-fructose diet, increased fatty acid input, type II diabetes, hyperlipidemia, obesity, and metabolic syndrome (which is characterized by at least three of the following five health problems: Central obesity, hypertension, hyperglycemia, elevated triglyceride levels in the blood, and decreased HDL lipid in serum) are all risk factors in the disease's pathogenesis[9–12]. The prevalence pattern, pathophysiology, clinical characteristics, and prognosis of metabolic syndrome and NAFLD are comparable[13]. NAFLD is linked to cardiovascular disease risk factors such as the thickening of the carotid arterial wall and decreased endothelial flow-mediated vasodilation. NAFLD increases mortality and predicts future cardiovascular disease (CVD) events[14]. Subsequently, numerous kinds of research have shown a link between NAFLD risk factors and socioeconomic characteristics[15,16].

Patients with NAFLD should change their lifestyles, such as food, physical activity, and weight. Hepatic steatosis can be improved by following a low-calorie diet and increasing physical activity[2]. A hypocaloric diet (a decrease of 500–1,000 calories per day) combined with moderate activity has the highest likelihood of long-term weight loss[17,18]. Steatosis seems to be improved by losing at least 3% to 5% of body weight, while most histological characteristics of NASH, including fibrosis, seem to be improved by losing more weight (7–10%)[19,20].

2. Material and Method

Materials

2.1 Questionnaire form

We used semi constructed questionnaire for data collection. The questionnaire form was sourced from; studies about risk factors of NAFLD associated with biochemical tests[21–24]. The World health organization (WHO) STEPwise approach to noncommunicable disease risk factor surveillance (STEPS) guideline is used for assessing smoking and physical activity[25].

The researcher used a questionnaire for data collection during face-to-face interviews. Personal and sociodemographic such as age, sex, marital status, level of education, and occupational role are included in the questionnaire. It also asked about smoking, nutrition, and physical activity.

The nutritional status of the Sulaimani population was assessed over time using a semi-quantitative food-frequency questionnaire (FFQ)[21–23,25,26]. Rice, bread, pasta, dairy products, red meat, chicken, fish, fruits, vegetables, eggs, sweets/desserts, fast food, sunflower, sweetened drinks (soda, juice), tea, and coffee were among the 16 food and food groups included in the FFQ, which was designed to the diet of the people of Sulaimani. Restaurant foods, such as burgers, pizza, and

shawarma, are classified as fast food. Milk, yogurt, and cheese are all examples of dairy products. Participants were asked to estimate how often they consumed certain meals in the six months preceding data collection by picking an option from the following four categories: never, daily, weekly, or monthly. Individuals who ingested an item 3 times daily ($3 \times 7 = 21$ times per week), five times per week ($5 \times 1 =$ five times per week), or three times per month ($3/4 = 0.75$ times per week) were assigned scores based on how often they consumed that item each week.

Blood samples were collected after a 12-hour overnight fast. The samples were then put through a Hitachi Cobas® c 311 automatic analyzer (Hitachi, Tokyo, Japan) at the Shar Hospital and Hitachi Cobas® 6000 (Cobas c 501) automatic analyzer (Hitachi, Tokyo, Japan) at the Baxshin Hospital to determine their HbA1c, alanine aminotransferase (ALT), s.triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), s.cholesterol and aspartate aminotransferase (AST).

Abdominal ultrasound was used to identify fatty liver using standardized guidelines [27–30]. The ultrasound was conducted on all individuals using both devices (PHILIPS Affiniti 30 and SIUI Ultrasound CTS 4000). The sonographic definition of fatty liver was based on a combination of liver-kidney contrast (bright liver) and vascular blurring. Sonographers blinded to the individuals' clinical and laboratory characteristics performed liver ultrasonography. To lower interference from stomach contents, participants fasted overnight for 8 hours. Ultrasonography of the liver was conducted in a quiet environment with moderate luminance.

2.2 Statistical analysis

Current study data was analyzed using SPSS (Statistical Package for the Social Sciences) version 22. Mean, standard deviation, frequency, and percentage are of the descriptive statistics that were calculated. The significance of the relationship between the categories was determined using a Chi-square test and Paired Samples T Test with a P-value of less than 0.05.

Method

2.3 Study design

This study was conducted as a case-control study targeting the Sulaimani city population attending the Shar Hospital (public sector) and Baxshin Hospital (private sector), carried out from February 2022 to July 2022. In this study, a questionnaire form was used to conduct interviews with the people who participated in the study. There were 148 people aged between 18 and 70 years who participated in the study. Out of them, 74 had NAFLD and were called "cases", The other 74 did not have liver disease and were called "controls". Inclusion criteria All patients aged 18 and 70 years were coming to the hospital for treatment. The exclusion criteria were alcoholics and aged more than 70 years. A convenience (non-probability) sampling method was used for data collection. After taking informed verbal consent from each, abdominal ultrasound was done to evaluate the liver state. After that, their blood was collected from intervenes that patient didn't eat food and fasted for 12 hours

3. Results and Discussions

3.1 Results

The findings in (Table 1) show that 74 individuals were diagnosed With NAFLD, and 74 participants were free from fatty liver. The majority of participants (35.6%) were aged between (30-44) years, (89.8%) of them were married, and 62.8 percent of respondents had a primary school degree in literacy. More than two third of the sample (80.5%) were from urban, and more than one-third of the sample (39.9%) had governmental employment. Finally, Results showed that 85.9% of participants were non-smokers, and 14.1% were smokers. In terms of participant characteristics, the case and control groups had no significant differences.

Table 2 provides information about comparing the case and control physical activity levels. The sufficiently active participant with NAFLD was recorded as having a higher rate of about 50%, while in control was approximately 35.1%. No significant differences were found when comparing the levels of physical activity in the case and control groups.

Except for Hba1c, all blood tests (s. cholesterol, s. triglyceride, HDL, LDL, ALT, AST) revealed significant differences between the mean values of patients with NAFLD and the control group ($p < 0.05$). Patients with NAFLD had higher mean S. triglyceride (295.50 ± 287.12 mg/dl), ALT, s. cholesterol, and LDL levels. However, when compared to the control group, mean HDL and AST levels in the patient group decreased. Regarding Hba1c, the difference between the patient and control was statistically insignificant, while the patient's mean was higher than the controls, as shown in Table(3).

This study showed that coffee, tea, and vegetables had statistically significant differences between cases and controls ($p < 0.05$). Coffee and tea were the most common food groups consumed by cases (24.01 ± 15.33) and controls (30.17 ± 16.17) compared to other food groups. Fast foods are the lowest food group consumed by cases (0.54 ± 1.22) and controls (0.40 ± 0.59). See table 4.

Results showed a significant difference ($p < 0.05$) in height, weight, and BMI measurement between patients and control groups. In contrast, those with NAFLD had a mean height, weight, and BMI greater than those in the control group (table 5).

The findings in table 6 reveal that more than half (55.4%) of patients with NAFLD were overweight compared to the control group (45.9%) and that (31.1%) of the control compared to cases (20.35%) had a normal BMI, the statistical significance of this difference ($p < 0.05$).

Figure 1 illustrates the smoking cigarettes status between the case and control. Results showed that 62% of participants were non-smokers, and 12% were smokers, suggesting that smoking cigarettes did not significantly affect patients with NAFLD compared to the control group.

3.2 Discussion

In current study discovered that the control group consumed more vegetables and drank more tea than the NAFLD patients. This difference was found to be statistically significant ($P < 0.05$) and a lower risk of developing NAFLD in the sulaimani population, supported by other studies that show significant differences between tea and vegetables with NAFLD [31–34]. Because vegetables and drinks, including coffee, black tea, green tea, and dark chocolate, all include polyphenols, which are natural phytochemical components. Approximately 8,000 polyphenolic compounds have been discovered in various plant species, each with its own fantastic set of advantageous properties and

biological activities, such as antioxidants[35], anti-inflammatory[36], anti-hyperglycemic effects[37,38], anti-allergic[39], anti-carcinogenic[40], anti-thrombotic[41], anti-hypertensive[42], anti-viral[43,44], and have been shown to improve lipid metabolism [32,33]. It may be since the people of the Kurdistan region usually drink tea after meals and eat vegetables with them. Studies in Baghdad, Iran, and Lebanon that disagreed with the current study demonstrated no significant relationship both tea and vegetables with NAFLD[45–47].

Most Sulaimani people's diets shown in table 4 did not seem to have any statistically significant link to NAFLD except tea and vegetables. It's possible that the participants' diets as a whole are to blame for their inability to form meaningful relationships with each other since they contain both healthy nutrients that may protect against NAFLD and unhealthy nutrients that may increase the risk of NAFLD. Red meat, chicken, fast food, whole-wheat bread, rice, fruits, soft drinks, sweets, juices, pickles, and salty foods are all common in Sulaimani diets and have all been linked to NAFLD risk[48–50]. There are also anti-inflammatory and antioxidant elements present. Curcumin, cinnamon, black tea, cardamom, black pepper, cloves, ginger, and onions[51–53], the most often used spices in Sulaimani cuisine, may have liver-protective properties.

This research also discovered a significant relationship between those who are overweight and NAFLD. NAFLD individuals had significantly higher mean weight and BMI than the control group. Because this region's population usually uses rice with bread as a main meal at least once a day and serves vegetables and snacks twice daily. This result, in line with other studies, showed a significant association between weight and BMI [54–56]. In contrast, other studies found no association between weight and NAFLD[57,58].

The current study regarding physical activity showed no statistically significant difference between patients and controls. Our findings disagree with studies showing a significant association between physical activity and NAFLD[59,60]. Regarding smoking, our study revealed no significant association between cases and controls. The study conducted in China agrees with my results[61]. However, it contradicts research that found smoking close correlated to NAFLD and has been known to cause oxidative stress[45,59,62].

Regarding biochemical parameters, people with NAFLD had higher mean s.triglyceride, LDL, and s.cholesterol levels and lower mean HDL values than those without NAFLD. Compared to the control group, there was a statistically significant difference in the lipid profile and NAFLD cases. This result agrees with research conducted in Puducherry, South India. According to the study, individuals with NAFLD had elevated levels of LDL and TG and a decreased HDL level, and the NAFLD group had a high level of dyslipidemia[63].

4. Conclusion

A higher intake of tea and vegetable had a good effect on people's health, decreasing NAFLD. There was no relationship between smoking and physical activity in NAFLD patients in Sulaimani City population.

5. Recommendation

We recommend the Kurdistan Regional Government educate the population about the disease, its dangers, and methods of prevention since the disease's prevalence is increasing dramatically due

to people's unhealthy lifestyles. People should do at least 150 minutes of moderate exercises, such as walking and cycling or 75 minutes of intense exercise, such as football and swimming. It is proven not for NAFLD but diabetes type 2, hypertension, and heart diseases are also helpful. Drinking plenty of low-sugar tea and eating vegetables and spices can protect them from liver disease because of some beneficial substances. Try to lose weight ,Overweight people are more likely to develop the disease.

References

- [1] Alsuhaibani K, Althunayyan F, Alsudays A, Alharbi A, Aljarallah B. Nonalcoholic fatty liver disease in lean and obese patients in Saudi patients from a single center. *J Family Med Prim Care* 2021;10:3381. https://doi.org/10.4103/jfmpc.jfmpc_185_21.
- [2] Chalasani N, Younossi Z, Lavine JE, Charlton M, Cusi K, Rinella M, et al. The diagnosis and management of nonalcoholic fatty liver disease: practice guidance from the American Association for the Study of Liver Diseases. *Hepatology* 2018;67:328–57.
- [3] Rinella ME, Sanyal AJ. Management of NAFLD: a stage-based approach. *Nat Rev Gastroenterol Hepatol* 2016;13:196–205.
- [4] Vernon G, Baranova A, Younossi ZM. Systematic review: the epidemiology and natural history of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis in adults. *Aliment Pharmacol Ther* 2011;34:274–85.
- [5] Kojima S, Watanabe N, Numata M, Ogawa T, Matsuzaki S. Increase in the prevalence of fatty liver in Japan over the past 12 years: analysis of clinical background. *J Gastroenterol* 2003;38:954–61.
- [6] Zhou Y-J, Li Y-Y, Nie Y-Q, Ma J-X, Lu L-G, Shi S-L, et al. Prevalence of fatty liver disease and its risk factors in the population of South China. *World Journal of Gastroenterology: WJG* 2007;13:6419.
- [7] Lankarani KB, Ghaffarpasand F, Mahmoodi M, Lotfi M, Zamiri N, Heydari ST, et al. Non alcoholic fatty liver disease in southern Iran: a population based study. *Hepat Mon* 2013;13.
- [8] Amirkalali B, Poustchi H, Keyvani H, Khansari MR, Ajdarkosh H, Maadi M, et al. Prevalence of non-alcoholic fatty liver disease and its predictors in north of Iran. *Iran J Public Health* 2014;43:1275.
- [9] Crabb DW, Galli A, Fischer M, You M. Molecular mechanisms of alcoholic fatty liver: role of peroxisome proliferator-activated receptor alpha. *Alcohol* 2004;34:35–8.
- [10] Machado M, Cortez-Pinto H. Non-alcoholic steatohepatitis and metabolic syndrome. *Curr Opin Clin Nutr Metab Care* 2006;9:637–42.
- [11] Angelico F, del Ben M, Conti R, Francioso S, Feole K, Maccioni D, et al. Non-alcoholic fatty liver syndrome: a hepatic consequence of common metabolic diseases. *J Gastroenterol Hepatol* 2003;18:588–94.
- [12] Juanola O, Martínez-López S, Francés R, Gómez-Hurtado I. Non-alcoholic fatty liver disease: Metabolic, genetic, epigenetic and environmental risk factors. *Int J Environ Res Public Health* 2021;18. <https://doi.org/10.3390/ijerph18105227>.
- [13] Loria P, Lonardo A, Carulli L, Verrone AM, Ricchi M, Lombardini S, et al. the metabolic syndrome and non-alcoholic fatty liver disease. *Aliment Pharmacol Ther* 2005;22:31–6.

- [14] Targher G, Arcaro G. Non-alcoholic fatty liver disease and increased risk of cardiovascular disease. *Atherosclerosis* 2007;191:235–40.
- [15] Santos AC, Ebrahim S, Barros H. Gender, socio-economic status and metabolic syndrome in middle-aged and old adults. *BMC Public Health* 2008;8:1–8.
- [16] Manuck SB, Phillips J, Gianaros PJ, Flory JD, Muldoon MF. Subjective socioeconomic status and presence of the metabolic syndrome in midlife community volunteers. *Psychosom Med* 2010;72:35.
- [17] Haufe S, Engeli S, Kast P, Böhnke J, Utz W, Haas V, et al. Randomized comparison of reduced fat and reduced carbohydrate hypocaloric diets on intrahepatic fat in overweight and obese human subjects. *Hepatology* 2011;53:1504–14. <https://doi.org/10.1002/HEP.24242>.
- [18] Kirk E, Reeds DN, Finck BN, Mayurranjan MS, Patterson BW, Klein S. Dietary fat and carbohydrates differentially alter insulin sensitivity during caloric restriction. *Gastroenterology* 2009;136:1552–60.
- [19] Musso G, Cassader M, Rosina F, Gambino R. Impact of current treatments on liver disease, glucose metabolism and cardiovascular risk in non-alcoholic fatty liver disease (NAFLD): A systematic review and meta-analysis of randomised trials. *Diabetologia* 2012;55:885–904. <https://doi.org/10.1007/S00125-011-2446-4>.
- [20] Vilar-Gomez E, Martinez-Perez Y, Calzadilla-Bertot L, Torres-Gonzalez A, Gra-Oramas B, Gonzalez-Fabian L, et al. Weight loss through lifestyle modification significantly reduces features of nonalcoholic steatohepatitis. *Gastroenterology* 2015;149:367–78.
- [21] Trovato FM, Martines GF, Catalano D. Addressing Western dietary pattern in obesity and NAFLD. *Nutrire* 2018;43. <https://doi.org/10.1186/s41110-018-0067-0>.
- [22] Mehdi Al Khalidi N, Ghanim Kadhim Z, Yahya Almousawi H. Dietary patterns in adult patients with Non-Alcoholic Fatty Liver Disease in Iraq 2021.
- [23] P. Kalafati I, Borsa D, Dimitriou M, Revenas K, Kokkinos A, v. Dedoussis G. Dietary patterns and non-alcoholic fatty liver disease in a Greek case–control study. *Nutrition* 2019;61:105–10. <https://doi.org/10.1016/j.nut.2018.10.032>.
- [24] H. MOHAMMED M, SH. ALDUHOKY L, A. SARHAN S, H. AHMED I. ASSOCIATION OF NON ALCOHOLIC FATTY LIVER WITH TYPE 2 DIABETES MELLITUS. *Duhok Medical Journal* 2021;15:11–22. <https://doi.org/10.31386/dmj.2021.15.1.2>.
- [25] Instrument HOWHOS. The WHO STEPwise approach to noncommunicable disease risk factor surveillance (STEPS). Geneva: World Health Organization 2016.
- [26] Majid M, Mutar MT, Ibrahim MJ, Saad A, Goyani MS, Abdulmortaefa A, et al. DEVELOPING A RELATIVELY VALIDATED AND REPRODUCIBLE FOOD FREQUENCY QUESTIONNAIRE IN BAGHDAD, IRAQ. *Global Journal of Public Health Medicine* 2022;4:51022.
- [27] Hamer OW, Aguirre DA, Casola G, Lavine JE, Woenckhaus M, Sirlin CB. Fatty liver: imaging patterns and pitfalls. *Radiographics* 2006;26:1637–53.
- [28] Quinn SF, Gosink BB. Characteristic sonographic signs of hepatic fatty infiltration. *American Journal of Roentgenology* 1985;145:753–5.
- [29] Mathiesen UL, Franzen LE, Åselius H, Resjö M, Jacobsson L, Foberg U, et al. Increased liver echogenicity at ultrasound examination reflects degree of steatosis but not of fibrosis in asymptomatic patients with mild/moderate abnormalities of liver transaminases. *Digestive and Liver Disease* 2002;34:516–22.

- [30] Trovato FM, Catalano D, Musumeci G, Trovato GM. 4Ps medicine of the fatty liver: the research model of predictive, preventive, personalized and participatory medicine—recommendations for facing obesity, fatty liver and fibrosis epidemics. *EPMA Journal* 2014;5:124.
- [31] Masterjohn C, Bruno RS. Therapeutic potential of green tea in nonalcoholic fatty liver disease. *Nutr Rev* 2012;70:41–56.
- [32] Rodriguez-Ramiro I, Vauzour D, Minihane AM. Polyphenols and non-alcoholic fatty liver disease: impact and mechanisms. *Proceedings of the Nutrition Society* 2016;75:47–60.
- [33] van de Wier B, Koek GH, Bast A, Haenen GRMM. The potential of flavonoids in the treatment of non-alcoholic fatty liver disease. *Crit Rev Food Sci Nutr* 2017;57:834–55.
- [34] Salomone F, Godos J, Zelber-Sagi S. Natural antioxidants for non-alcoholic fatty liver disease: molecular targets and clinical perspectives. *Liver International* 2016;36:5–20.
- [35] Hakim IA, Harris RB, Brown S, Chow HHS, Wiseman S, Agarwal S, et al. Effect of increased tea consumption on oxidative DNA damage among smokers: a randomized controlled study. *J Nutr* 2003;133:3303S-3309S.
- [36] Hodges JK, Sasaki GY, Bruno RS. Anti-inflammatory activities of green tea catechins along the gut–liver axis in nonalcoholic fatty liver disease: Lessons learned from preclinical and human studies. *J Nutr Biochem* 2020;85:108478.
- [37] Wolfram S. Effects of green tea and EGCG on cardiovascular and metabolic health. *J Am Coll Nutr* 2007;26:373S-388S.
- [38] Ueda M, Nishiumi S, Nagayasu H, Fukuda I, Yoshida K, Ashida H. Epigallocatechin gallate promotes GLUT4 translocation in skeletal muscle. *Biochem Biophys Res Commun* 2008;377:28690.
- [39] Maeda-Yamamoto M, Inagaki N, Kitaura J, Chikumoto T, Kawahara H, Kawakami Y, et al. O-methylated catechins from tea leaves inhibit multiple protein kinases in mast cells. *The Journal of Immunology* 2004;172:4486–92.
- [40] Khan N, Mukhtar H. Multitargeted therapy of cancer by green tea polyphenols. *Cancer Lett* 2008;269:269–80.
- [41] Stangl V, Lorenz M, Stangl K. The role of tea and tea flavonoids in cardiovascular health. *Mol Nutr Food Res* 2006;50:218–28.
- [42] Kim J, Formoso G, Li Y, Potenza MA, Marasciulo FL, Montagnani M, et al. Epigallocatechin gallate, a green tea polyphenol, mediates NO-dependent vasodilation using signaling pathways in vascular endothelium requiring reactive oxygen species and Fyn. *Journal of Biological Chemistry* 2007;282:13736–45.
- [43] Hamza A, Zhan C-G. How can (–)-epigallocatechin gallate from green tea prevent HIV-1 infection? Mechanistic insights from computational modeling and the implication for rational design of anti-HIV-1 entry inhibitors. *J Phys Chem B* 2006;110:2910–7.
- [44] Xu J, Wang J, Deng F, Hu Z, Wang H. Green tea extract and its major component epigallocatechin gallate inhibits hepatitis B virus in vitro. *Antiviral Res* 2008;78:242–9.
- [45] al Khalidi NM, Kadhim ZG, Almousawi HY. Dietary patterns in adult patients with Non-Alcoholic Fatty Liver Disease in Iraq 2021.
- [46] Fakhoury-Sayegh N, Younes H, Heraoui GNHA, Sayegh R. Nutritional profile and dietary patterns of lebanese non-alcoholic fatty liver disease patients: a case-control study. *Nutrients* 2017;9:1245.

- [47] al Khalidi NM, Kadhim ZG, Almousawi HY. Dietary patterns in adult patients with Non-Alcoholic Fatty Liver Disease in Iraq 2021.
- [48] Vancells Lujan P, Viñas Esmel E, Sacanella Meseguer E. Overview of non-alcoholic fatty liver disease (NAFLD) and the role of sugary food consumption and other dietary components in its development. *Nutrients* 2021;13:1442.
- [49] Shen X, Jin C, Wu Y, Zhang Y, Wang X, Huang W, et al. Prospective study of perceived dietary salt intake and the risk of non-alcoholic fatty liver disease. *Journal of Human Nutrition and Dietetics* 2019;32:802–9.
- [50] Zhang S, Gu Y, Bian S, Górska MJ, Zhang Q, Liu L, et al. Dietary patterns and risk of non-alcoholic fatty liver disease in adults: a prospective cohort study. *Clinical Nutrition* 2021;40:537382.
- [51] Sahebkar A. Potential efficacy of ginger as a natural supplement for nonalcoholic fatty liver disease. *World Journal of Gastroenterology: WJG* 2011;17:271.
- [52] Mansour-Ghanaei F, Pourmasoumi M, Hadi A, Joukar F. Efficacy of curcumin/turmeric on liver enzymes in patients with non-alcoholic fatty liver disease: a systematic review of randomized controlled trials. *Integr Med Res* 2019;8:57–61.
- [53] Hajimonfarednejad M, Ostovar M, Raei MJ, Hashempur MH, Mayer JG, Heydari M. Cinnamon: A systematic review of adverse events. *Clinical Nutrition* 2019;38:594–602.
- [54] Fakhoury-Sayegh N, Younes H, Heraoui GNHA, Sayegh R. Nutritional profile and dietary patterns of lebanese non-alcoholic fatty liver disease patients: a case-control study. *Nutrients* 2017;9:1245.
- [55] Salehi-Sahlabadi A, Sadat S, Beigrezaei S, Pourmasomi M, Feizi A, Ghiasvand R, et al. Dietary patterns and risk of non-alcoholic fatty liver disease. *BMC Gastroenterol* 2021;21:1–12.
- [56] Lankarani KB, Ghaffarpasand F, Mahmoodi M, Lotfi M, Zamiri N, Heydari ST, et al. Non alcoholic fatty liver disease in southern Iran: a population based study. *Hepat Mon* 2013;13.
- [57] Tajima R, Kimura T, Enomoto A, Saito A, Kobayashi S, Masuda K, et al. No association between fruits or vegetables and non-alcoholic fatty liver disease in middle-aged men and women. *Nutrition* 2019;61:119–24.
- [58] Liu C. Prevalence and risk factors for non-alcoholic fatty liver disease in Asian people who are not obese. *J Gastroenterol Hepatol* 2012;27:1555–60.
- [59] Salehi-Sahlabadi A, Sadat S, Beigrezaei S, Pourmasomi M, Feizi A, Ghiasvand R, et al. Dietary patterns and risk of non-alcoholic fatty liver disease. *BMC Gastroenterol* 2021;21:1–12.
- [60] Fakhoury-Sayegh N, Younes H, Heraoui GNHA, Sayegh R. Nutritional profile and dietary patterns of lebanese non-alcoholic fatty liver disease patients: a case-control study. *Nutrients* 2017;9:1245.
- [61] Chavez-Tapia NC, Lizardi-Cervera J, Perez-Bautista O, Ramos-Ostos MH, Uribe M. Smoking is not associated with nonalcoholic fatty liver disease. *World Journal of Gastroenterology: WJG* 2006;12:5196.
- [62] Han AL. Association between non-alcoholic fatty liver disease and dietary habits, stress, and health-related quality of life in Korean adults. *Nutrients* 2020;12:1555.
- [63] Santhoshakumari TMJ, Radhika G, Kanagavalli P. A study of anthropometric and lipid profile parameters in non-alcoholic fatty liver disease patients attending a tertiary care hospital at puducherry. *IOSR J Dent Med Sci (IOSR-JDMS)* 2017;16:33–7

Table 1: Characteristics Of Study Participants.

Participants Characteristics		Case <i>Freq.</i> (%) (N=74)	Control <i>Freq. (%)</i> (N=74)	Total <i>Freq. (%)</i> N=148	P Value
Age	18- 29	5 (6.8)	16 (21.6)	21(14.2)	0.069
	30- 44	14 (18.9)	39 (52.7)	53(35.8)	
	45- 59	29 (39.2)	8 (10.8)	37(25)	
	60- 69	26 (35.1)	11 (14.9)	37(25)	
	Mean Of The Case Age = 47± 1.2, And Mean Of The Control Age=32± 1.12				
Gender	Male	33 (44.6)	30 (40.5)	63(42.6)	0.440
	Female	41 (55.4)	44 (59.5)	85(57.4)	
Marital Statues	Single	3 (4.1)	9 (12.2)	12(8.2)	0.805
	Married	69 (93.2)	64 (86.5)	133(89.8)	
	Widowed	2 (2.7)	1 (1.4)	3(2)	
Level Of Education	Illiterate	17 (23.0)	21 (28.4)	38(25.7)	0.487
	Primary	28 (37.8)	26 (35.1)	54(36.5)	
	Secondary	14 (18.9)	11 (14.9)	25(16.9)	
	Diploma	13 (17.6)	6 (8.1)	19(12.8)	
	Bachelor	2 (2.7)	10 (13.5)	12(8.1)	
Residential Area	Urban	66 (89.2)	53 (71.6)	119(80.5)	0.938
	Suburban	6 (8.1)	20 (27.0)	26(17.5)	
	Rural	2 (2.7)	1 (1.4)	3(2)	
Employment Statues	Governmental Employment	22 (29.7)	25 (33.8)	47(31.7)	0.637
	Nongovernmental Employment	7 (9.5)	12 (16.2)	19(12.8)	
	Housewife	28 (37.8)	31 (41.9)	59(39.9)	
	Retired	15 (20.3)	5 (6.8)	20(13.6)	
	Unemployed (Unable To Work)	2 (2.7)	1 (1.4)	3(2)	
Economic State	Poor	19 (25.7)	6 (8.1)	25(16.9)	0.732
	Medium	47 (63.5)	48 (64.9)	95(64.2)	
	Good	8 (10.8)	20 (27.0)	28(18.9)	
Smoking State	Smoker	12(16.2)	9 (12.2)	21 (14.1)	0.6.2
	Non-Smoker	62(83.8)	65 (87.8)	127 (85.9)	
Note/ Data Presented As (Freq.=Frequency, N=Number Of Cases), Test Done By Chi-Square					

Table 2: Shows Levels Of Physical Activity In The Patient And Control.

Physical Activity	Case Freq. (%) (N=74)	Control Freq. (%) (N=74)	P Value
Sedentary Or Inactive	22(29.7)	24(32.4)	0.096
Insufficiently Active	15(20.3)	24(32.4)	
Sufficiently Active	37(50)	26(35.1)	
Note/ Data Presented As (Freq.=Frequency, N=Number Of Cases), Test Done By Chi-Square			

Table 3: Shows Serum Level Of Biochemical Parameters In Patients And Control Group.

Biochemical Parameters	Case Mean±SD	Control Mean±SD	P Value
Triglyceride	295.50±287.12	108.80±28.4	0.001
Cholesterol	167.89±37.60	156.76±26.96	<0.001
Hdl	44.32±18.77	50.16±20.32	<0.001
Ldl	109.05±36.78	100.32±18.89	0.013
Alt(Gpt)	26.41±9.01	25.43±15.30	0.039
Ast(Got)	22.95±7.57	26.22±12.19	0.001
Hba1c	6.70±1.73	5.61±0.89	0.118
Note/ data presented as mean±SD, Test done by Paired Samples T test			

Table 4: Shows The Distribution Of Daily Food Portions According To NAFLD.

Food Items	Case Mean±SD	Control Mean±SD	P Value
Fruits	9.22±5.52	10.45±5.04	0.095
Vegetables	6.45±4.64	8.05±4.56	0.042
Milk & Milk Products	11.02±5.14	11.13±4.01	0.149
Meat	2.48±3.11	1.32±1.38	0.087
Bread	13.62±8.29	11.71±6.10	0.157
Sweets/Desserts	2.75±3.71	1.87±1.82	0.293
Fast Food	0.54±1.22	0.40±0.59	0.052
Coffee/Tea	24.01±15.33	30.17±16.17	0.003
Sweetened Drinks (Soda, Juice)	1.67±2.25	2.38±3.07	0.698
Eggs	1.95±1.62	2.93±1.99	0.310
Fish	0.72±0.41	0.41±0.50	0.932
Chicken	3.2±2.10	3.00±2.12	0.855
Rice	9.33±4.48	7.53±4.40	0.785
Pasta	0.76±2.24	0.29±0.88	0.750
Sunflower	3.48±4.38	2.44±2.37	0.906
Note/ Data Presented As Mean±SD, Test Done By Paired Samples T Test			

Table 5: Distribution Of Anthropometric Measures According To NAFLD

Variables	Case Mean±SD	Control Mean±SD	P Value
Weight	78.97±10.95	75.05±11.97	<0.001
Height	168.24±7.91	167.36±8.07	0.01
Bmi	27.92±3.66	26.91±4.62	<0.001
Note/ Data Presented As Mean±SD, Test Done By Paired Samples T Test			

Table 6: Comparison Between BMI Of Case And Control

Bmi	Case Freq. (%) (N=74)	Control Freq. (%) (N=74)	TotalFreq. (%) (N=74)	P Value
Underweight		0 (0)	1 (1.4)	1(0.7)
Normal		15 (20.3)	23 (31.1)	38(25.8)
Overweight		41 (55.4)	34 (45.9)	75(50.6)
Obese		18 (24.3)	16 (21.6)	34(22.9)

Note/ Data Presented As (Freq.=Frequency, N=Number Of Cases), Test Done By Chi-Square